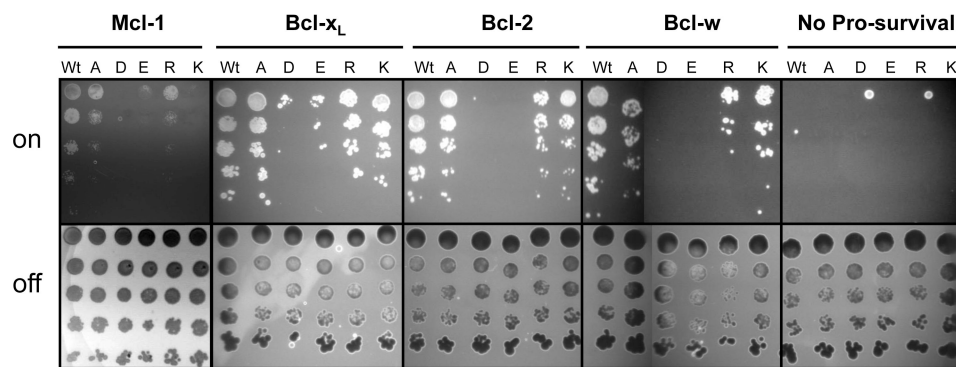


Supplemental Figures

Mutation to Bax beyond the BH3 domain disrupts interactions with pro-survival proteins and promotes apoptosis

Peter E. Czabotar, Erinna F. Lee, Geoff V. Thompson, Ahmad Z. Wardak, W. Douglas Fairlie, and Peter M. Colman

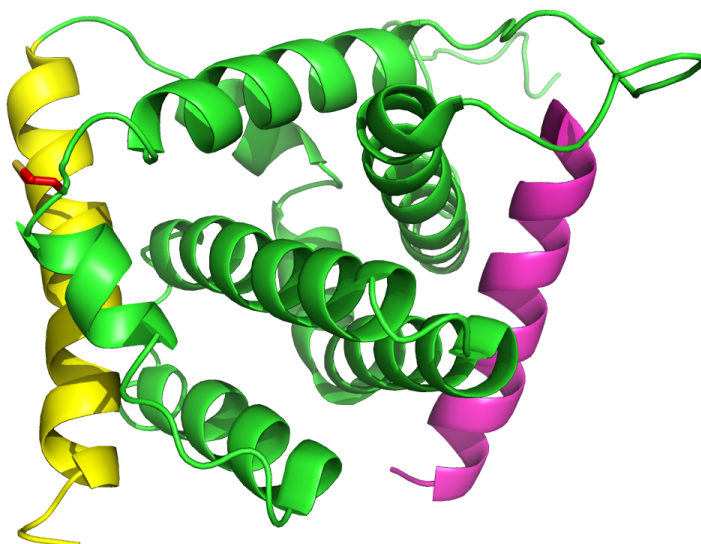
Supplemental Figure 1



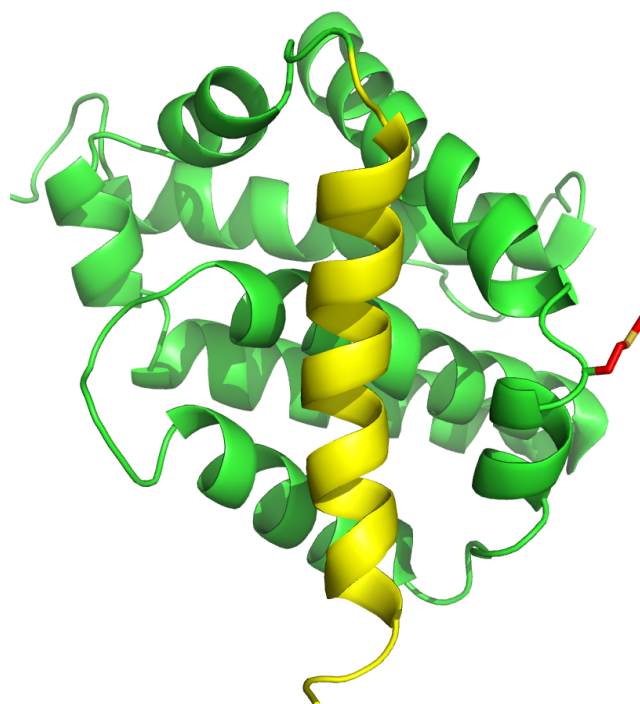
Supplemental Fig. 1. Bax wildtype and M74 mutants kill yeast cells. Rescue by pro-survival proteins fails in the case of the M74 mutants. Increasing dilutions of yeast cultures expressing both pro-survival proteins and Bax M74 mutants were spotted onto Galactose plates (induction on) or glucose plates (induction off). Labels across the top indicate the Bax mutation at position 74.

Supplemental Figure 2

A



B



Supplemental Fig. 2. (A) Bax M74 (red) is distal from the proposed Bim (magenta) binding site on the back side of Bax. (B) Residue M74 projects into solvent and is unlikely to influence binding of ligands in the canonical binding groove which in inactive Bax is accommodated by the hydrophobic tail (yellow).